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Reversion of tumorigenicity and decreased agarose clonability after EBV conversion of an IgH/myc translocation-carrying BL line.

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The Epstein-Barr virus (EBV)-negative Burkitt lymphoma (BL) line BL-41, and 5 independently established EBV-converted sublines, derived by infection with a transforming (B95-8) or a nontransforming (P3HR1) strain of EBV, were compared for clonability in semi-solid agarose and for tumorigenicity in immuno-suppressed mice. One P3HR1 viral convertant and 3 out of 4 B95-8 virus-converted sublines had a high (greater than 40%) agarose clonability, like the BL 41 parent, and were slightly more tumorigenic than BL-41. In contrast, the fourth B95-8 converted subline, BL-41/95, was virtually non-tumorigenic and its agarose clonability was much lower (3-23%). It showed a more drastic shift towards an LCL-like phenotype than the other convertants as reflected by high HLA class-I and EBV-encoded latent membrane protein (LMP) expression. BL 41/95 still contains the 8;14 IgH/myc translocation, carried by the parental line, and maintains the same relatively high steady-state level of c-myc mRNA and protein as the highly tumorigenic convertants. We conclude that the tumorigenicity of BL41/95 has been suppressed by a gene that acts at a level beyond the expression of the activated oncogene, in the same way as the revertants isolated from ras and SV-40-transformed cultures (Klein, 1987b; Bassin and Noda, 1987).

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